



## Quantification of myocardial delayed enhancement and wall thickness in hypertrophic cardiomyopathy: Multidetector computed tomography versus magnetic resonance imaging

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### ARTICLE INFO

#### Article history:

Received 17 December 2013

Received in revised form 23 May 2014

Accepted 27 May 2014

#### Keywords:

Hypertrophic cardiomyopathy  
Myocardial delayed enhancement  
MDCT  
MRI

### ABSTRACT

**Objectives:** To evaluate the accuracy of multidetector computed tomography (MDCT) in assessing myocardial delayed enhancement and left ventricle wall thickness in hypertrophic cardiomyopathy (HCM) compared with cardiac magnetic resonance (CMR) as the reference standard.

**Materials and methods:** Eighty consecutive patients (59 male;  $53.2 \pm 13.0$  years) were examined with MDCT, followed by CMR 1 day later. Cardiac CT angiography and a delayed CT were performed. CMR was performed according to a standardized protocol. Left ventricle wall thickness and positions of myocardial delayed enhancement were identified in both CMR and CT images according to the American Heart Association left ventricle 17-segment model. Myocardial delayed enhancement was characterized as “dense” (areas with clear defined borders) or “diffuse” and then quantified using both techniques.

**Results:** Left ventricle wall thickness determined by MDCT was significantly correlated with CMR ( $R = 0.88$ ,  $P < 0.01$ ). Compared with CMR, MDCT accurately diagnosed 74 of 78 (94.9%) patients and 1243 of 1326 (93.7%) segments. For dense myocardial delayed enhancement, MDCT significantly correlated with CMR ( $R = 0.88$ ,  $P < 0.01$ ) and slightly underestimated myocardial delayed enhancement (mean,  $-3.85\%$ ; lower and upper limits of agreement,  $-13.40\%$  and  $5.70\%$ , respectively).

**Conclusions:** MDCT provides reliable quantification of myocardial delayed enhancement and evaluation of left ventricle wall thickness and has a good correlation with CMR in patients with HCM when a comprehensive cardiac CT protocol is used and can be applied for intervention planning.

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### 1. Introduction

Multidetector computed tomography (MDCT) can assess coronary artery and global and regional myocardial function [1]. Studies have demonstrated that delayed enhancement MDCT would be a reliable technique for assessing myocardial viability as shown in patients after myocardial infarction [2–6] due to contrast media accumulation within infarcted regions. However, literature reporting on the performance of CT myocardial delayed enhancement (MDE) in hypertrophic cardiomyopathy (HCM) is rare [7,8].

**Abbreviations:** AHA, American Heart Association; BMI, body mass index; CMR, cardiovascular magnetic resonance; CNR, contrast to noise ratio; CT, computed tomography; HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter-defibrillator; LV, left ventricle; MDCT, multidetector computed tomography; MDE, myocardial delayed enhancement; SD, standard deviation; SI, signal intensity.

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In patients with HCM, the extent of MDE and precise anatomical assessment of the left ventricle (LV) are important parameters for the planning of surgical septal myectomy [9]. Cardiac CT, including both CT angiography and MDE imaging, would offer a non-invasive exam in patients with HCM who are scheduled for interventional procedures. Patients with HCM usually suffered from arrhythmia or have a cardiac pacemaker, in which case cardiac magnetic resonance (CMR) is contraindicated. Without regard to coronary artery, MDCT can image the cardiac chamber structure even in patients with arrhythmia, and variety of image reconstruction algorithm can partially eliminate the metal devices artifacts. Preliminary results have showed the feasibility of using CT to display HCM [7,8], but no study with a large number of cases has yet validated the quantification of MDE and characterization of the MDE subtype.

Therefore, the purpose of our study was to prospectively compare MDCT and CMR imaging for characterizing myocardial fibrosis (“dense” versus “diffuse” type) and performing a

quantitative assessment of MDE in HCM as well as evaluating myocardial wall thickness.

## 2. Materials and methods

### 2.1. Patients

The study was approved by Beijing Anzhen Hospital Ethics Committee. Informed written consent was obtained from each patient. A detailed explanation concerning radiation dose exposure and the risks related to CT and CMR was given to each patient.

#### 2.1.1. Inclusion criteria

From August 2011 to December 2012, a total of 80 patients with HCM were prospectively enrolled. The diagnosis of HCM was based on standardized criteria of LV hypertrophy ( $\geq 15$  mm or  $\geq 13$  mm in documented family disease) not originating from other causes determined by echocardiography according to the American Heart Association (AHA) guidelines for the diagnosis of HCM [10].

#### 2.1.2. Exclusion criteria

Patients with a history of myocardial infarction were excluded. Subjects with contraindications for CMR (claustrophobia, metal implants, cardiac pacemakers etc.) or MDCT (pregnancy, impaired renal function, contrast media allergy) were excluded. All patients received CMR and MDCT within a 2-day period.

### 2.2. CT protocol

The cardiac CT examination was performed using a dual-source CT (DSCT) scanner (Somatom Definition Flash; Siemens Healthcare, Erlangen, Germany). First, arterial phase imaging was applied with retrospective electrocardiography (ECG) gating using a spiral technique. The scanning parameters were as follows: detector collimation,  $2 \times 64 \times 0.6$  mm; rotation time, 280 ms; and temporal

resolution,  $>83$  ms. Tube voltage (kV) and tube current were chosen based on the patient's body habitus: 120 kV was used for patients with a body mass index (BMI)  $\geq 25$  kg/m<sup>2</sup>, 100 kV was used for those with a BMI  $<25$  kg/m<sup>2</sup>; and the tube current was 330–410 mAs. Tube current modulation and pitch adaptation were used. A total of 90 mL of iodinated contrast media (Ultravist 370 mgI/mL; Bayer Schering Healthcare, Berlin, Germany) was administered intravenously.

For MDE, a second CT scan with step-and-shoot prospective ECG triggering at 75% of the R-R interval was performed 7 min after the arterial phase contrast media injection. Tube current was 362 mAs. Tube voltage was adapted the patient's BMI: 100 kV for patients with a BMI  $\geq 25$  kg/m<sup>2</sup> and 80 kV for those with a BMI  $<25$  kg/m<sup>2</sup>. MDE images were reconstructed using a soft kernel (B10f) and reformatted into serial 8 mm-thick multiplanar reformation images (serial short-axis and two- and four-chamber long-axis slices).

The total radiation dose for both scans was estimated by multiplying the dose-length product by the conversion coefficient ( $0.014 \text{ mSv} \times \text{Gy}^{-1} \times \text{cm}^{-1}$ ) [11].

### 2.3. CMR acquisition

CMR imaging was performed using a 1.5 T MRI unit (Sonata; Siemens Healthcare, Erlangen, Germany) with a 12-element phased array cardiac coil. After scout CMR images were obtained, balanced steady state free precession cine images were acquired in horizontal and vertical long-axis views, while short-axis views were obtained parallel to the atrioventricular groove and included the entire LV. The imaging parameters were as follows: repetition/echo time, 41.28/1.51 ms; image matrix,  $224 \times 256$ ; flip angle,  $50^\circ$ ; field of view,  $340 \times 280$ ; and band width, 977 Hz.

MDE images in the same orientations as the cine images were acquired 10 min after the intravenous infusion of gadolinium chelate contrast media (Magnevist, 0.2 m mol/kg; Bayer Schering

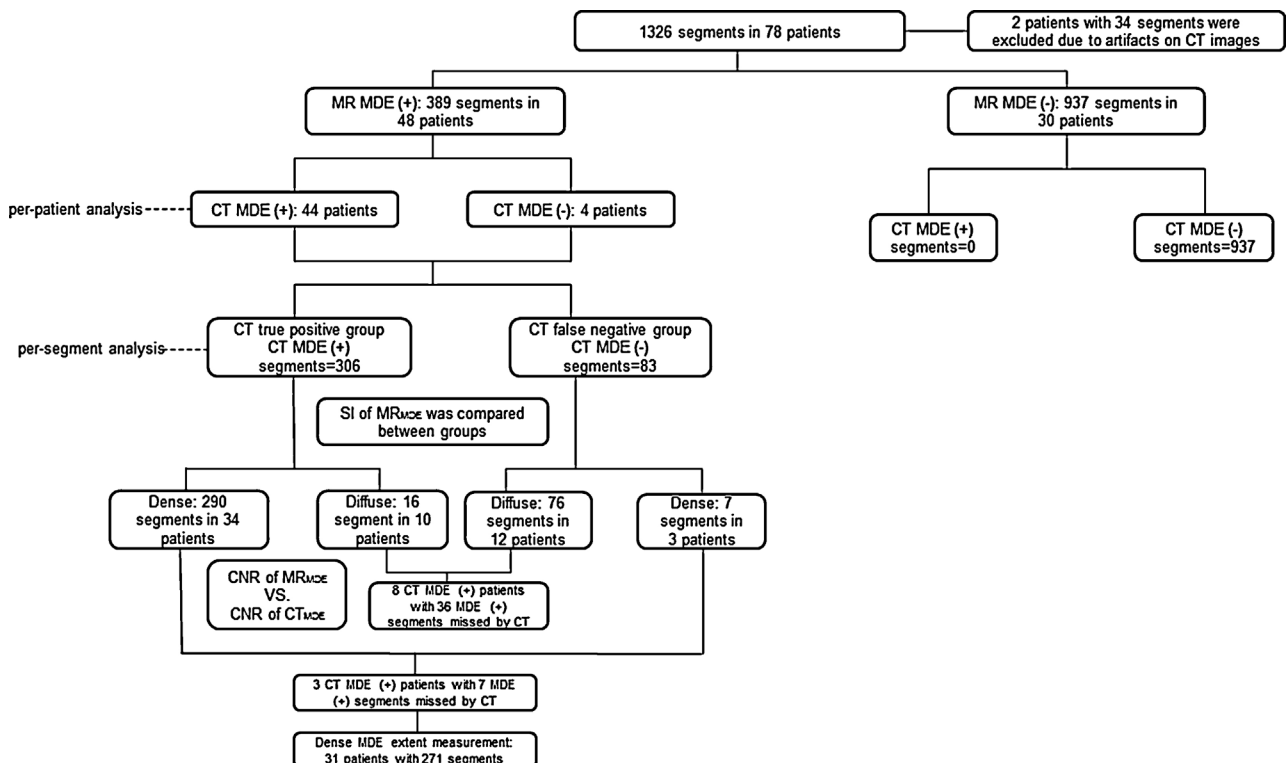


Fig. 1. Flow chart of myocardial delayed enhancement image analysis.

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